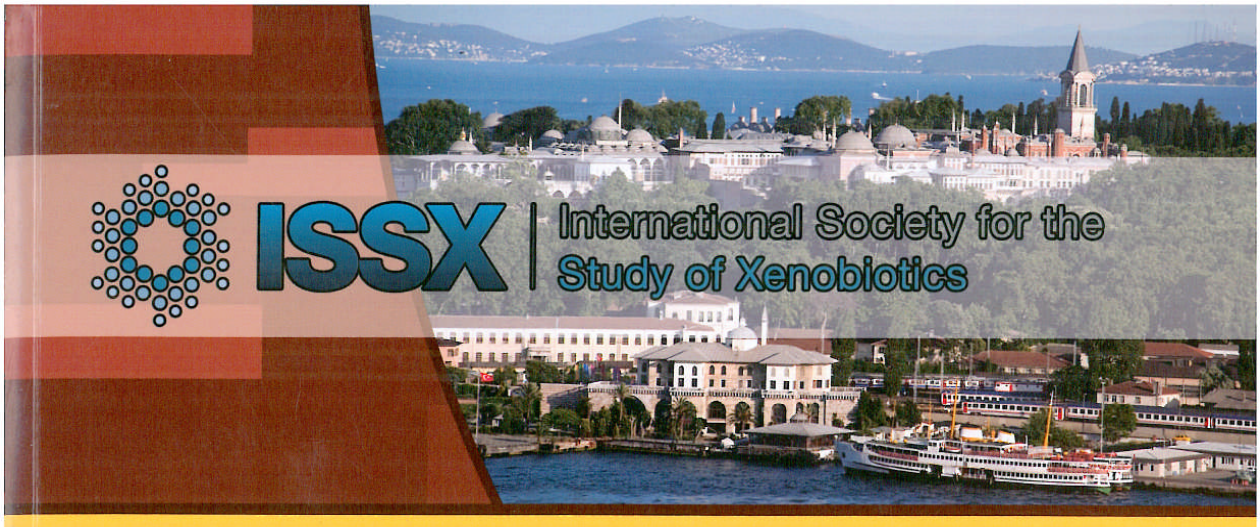

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PROGRAM & ABSTRACTS

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TÜBA

P166. PREVENTIVE EFFECT OF MAGNESIUM ON NICKEL HEPATOTOXICITY AND NEPHROTOXICITY IN ALBINO (WISTAR) RATS

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We studied the effect of intraperitonial magnesium treatment on nickel sulphate-induced hepatotoxicity and nephrotoxicity in Wistar strain male albino rats. Liver and kidney dysfunction parameters represented by aspartate transaminase (AST), alanine transaminase (ALT), alkaline phosphatase (ALP), blood glucose, serum total protein, serum urea, serum creatinine, and serum belurebin were estimated. Liver glutathione level, catalase and GPx activities were also determined in liver as indicators of oxidative damage. Nickel treatment was led to high serum. glucose concentration and produced hepatotoxicity and nephrotoxicity characterized by increasing GPT, GOT and alkaline phosphatase activities, serum total protein, serum urea, serum creatinine and serum belurebin concentrations. In addition liver glutathione level, catalase and GSH-Px activities were diminished due to high lipid peroxidation. The simultaneous administration of magnesium with nickel sulphate resulted in a remarkable improvement of the previous parameters comparison with rats treated with nickel alone. Normal control and magnesium treated animals revealed normal histology of liver. On the other hand, nickel treated animals showed alterations in normal hepatic histoarchitecture which comprise of vacuolization of the hepatocytes and dilatation of sinusoids. Administration of magnesium to nickel treated rats resulted in marked improvement in the structure of hepatocytes, thus emphasizing the protective potential of magnesium in restoring the altered hepatic histoarchitecture. In conclusion, nickel sulphate led to liver and kidney dysfunctions and hepatic lipid peroxidation in animals, but simultaneous treatment with magnesium offers a relative protection against nickel induced hepatotoxicity, nephrotoxicity and lipid peroxidation.

P167. PROTECTIVE ROLE OF ASCORBIC ACID ON LAMBDACYHALOTHRIN-INDUCED OXIDATIVE STRESS AND ALTERATIONS OF ACETYLCHOLINESTERASE ACTIVITY IN THE CEREBELLUM OF ADULT RATS

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The wide use and wide-spectrum toxicity of synthetic pyrethroids (SPs) insecticides make them an emerging ecotoxicological concern. The objective of the current study was to investigate the involvement of oxidative stress on lambda-cyhalothrin (LTC)-induced cerebellum damages in adult rats, and to evaluate the possible protective effect of vitamin C (Vit. C) as antioxidant. Exposure rats to lambda-cyhalothrin during 3 weeks, caused a significant ($P < 0.01$) increase in the levels of LPO, nitric oxide (NO) and protein carbonyls (PCO) along with significant ($P < 0.01$) decrease in the levels of reduced glutathione (GSH) and the activities of AChE, superoxide dismutase, catalase, glutathione peroxidase, glutathione reductase and gluthione-*S*-transeferase, ($P < 0.05$) compared to control group. Oral administration of vitamin C (200 mg/kg/day) to LTC-treated rats significantly ($P < 0.01$) reduced the levels of LPO, nitric oxide and protein carbonyls and increased the activities of GSH and antioxidant enzyme activities. Our results indicated that vitamin C attenuated the lipid peroxidation, protein oxidation and impaired antioxidant enzyme activities in LTC exposed rats. Thus suggested that ascorbic acid protected the brain against toxic effects of synthetic pyrethroids.

P168. STUDY OF OCCUPATIONNAL HAZARDS ON HUMAN INFERTILITY

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Research into occupational exposures and effects on reproductive systems has made important scientific contributions in the past few decades. In the workplace, sources of exposure to these xenobiotics are numerous because they are very different products (pesticides, solvents, metals ....), and toxicity is mainly long term because the absorption is at small doses over long periods. This study was conducted in the laboratory of Cytogenetics-Ibn Sina Annaba, (ALGERIA), had a goal of highlighting the impact of pesticides, metals, organic solvents and construction materials, in male fertility. On 2350 spermiograms, over a period of 05 years, we have identified a target population of 270 workers in atmospheres chemically polluted. The results show a slur of all subjects in six groups: workers in masonry, painting, carpentry, agriculture, plumbing, mechanics and employees ISPAT (major steel manufacturing plant in the north east of Algeria).

P169. ABSTRACT WITHDRAWN

P170. THE EPIDEMIOLOGYIC STUDY OF SUICIDE IN NORTH WEST OF IRAN

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The Epidemiologyic Study of Suicide in Ardabil Province BetweenYear 2003-2009. Objective: Suicide is an important problem for social safety and health . Recognizing some factors of risk forecasting after epidemiologic studies on people whom attempted to suicide could prepare and present outlines and proper guides for preventing by health and social planners. this research is purposed on suicide epidemiologic study to obtain full and enough data about

deceased people by suicide has been accomplished in Ardebil province. Methods: The accomplished research is a descriptive and analytic sectional research which is based on all deceased people by suicide during 6 years, which their statistics have been registered in Bualy, Fatemi & Alavy hospitals and forensic medicine of Ardebil province. The used tool was created researcher form that included epidemiologic particular of deceased people. The data were analyzed using SPSS software version 13. Results: The highest rate of suicide had been belonged to age rank of 15-24 years(43.8%) and had occurred in female (62.5, %), married people (57.8%) urban society (65.6%) and patient without past chronic physical illness or psychiatric history and without pre-attempt suicide. Self poisoning by drugs and toxins was the most common used method in this case (90.6%). Orderly among drugs and toxins organo-phosphorate toxin, tricyclic antidepressants and aluminum phosphid had been most used. Conclusion: This research is indicating various risk factors such as sex, age, marriage status, residing place of society and easy access to drugs and toxins. According to obtained results, it is better or proper to accomplished related education and training of preventation and interference of mental health in second and third decades of life with allocation of much time for females (especially the married people of this group). It is suggested that must be considered more attention to education, preventation, diagnosis and treatment of poisoning especially poisoning by most common Drugs and toxins.

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P171. THE IMPACT OF PROPICONAZOLE ON THE FERTILITY IN DOMESTIC RABBITS

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The main objective of this work was to study the effect of a widely used fungicide Propiconazole on fertility in the rabbits *Oryctologus cuniculus.* The treatment was made in food with two doses 1 and 5 mg/ml/kg of the food /day for 15 days. Summarizing the finding, it can be established that the administration of Propiconazole in the treated groups as compared with control groups caused a reduction in the weight of testes with increase in the weight of the liver. The present experiment reported a very significant reduction in the number, vitality and mobility in the treated spermatozoa. In conclusion, Administration of propiconazole affects the biology of sperms.

P172. THE MECHANISM OF DEFECTS IN SEXUAL BEHAVIOR BY MATERNAL EXPOSURE TO DIOXIN: FOCUSING ON GENE EXPRESSION IN THE PITUITARY AND HYPOTHALAMUS

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Many forms of reproductive toxicity, such as defects in sexual behavior caused by 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) occur in pups whose mother is exposed to the lower doses of this substance. However, the mechanism underlying these defects remains to be clarified in spite of many researches conducted so far. Our previous studies have provided evidence that the administration of TCDD (1 μg/kg, orally) to pregnant Wistar rats at gestational day 15 (GD15) causes a reduction in gonadotropin biosynthesis in the fetal pituitary, resulting in the attenuated expression of steroidogenic proteins in the fetal testes. Such attenuation occurred during a short period ranging from GD20 to postnatal day 0 (PND0). The direct supplementation of equine chorionic gonadotropin into the fetuses exposed to TCDD at GD15 restored not only the reduced expression of gonadal steroidogenic proteins but also defects in sexual behavior. This observation strongly supports the view that defects in gonadotropin formation trigger the impaired expression of steroidogenic proteins. To further clarify the mechanism of TCDD effect on fetal gonadotropin biosynthesis, we investigated whether TCDD can directly affect the fetal pituitary to reduce gonadotropin biosynthesis, using cultured pituitary. When fetal pituitary was cultured in the presence of gonadotropin-releasing hormone (GnRH), TCDD interfered with the GnRH-induced expression of gonadotropin β-subunit. Such an interference was fetus-specific because cultured postnatal pituitary (PND7) was insensitive to TCDD treatment. These observations suggest that TCDD reduces gonadotropin biosynthesis via, at least partially, its direct action on the fetal pituitary. We then performed DNA microarray analysis to identify the target genes linked to a reduction in gonadotropin β-subunit (fetus) and to defects in sexual behavior (adult). In fetal pituitary, a number of genes, such as transcription factors and apoptosis-related factors, as well as gonadotropin β-subunit were affected by maternal exposure to TCDD at GD15 (1.3-fold up: 86 genes, 0.7-fold down: 59 genes). The effect of TCDD was also observed in fetal hypothalamus, the regulatory organ of the pituitary. For example, the expression of some growth factors and signal transduction factors was reduced by TCDD. These results suggest that TCDD damages the gene regulation of fetal pituitary and hypothalamus, and this is a possible mechanism whereby the attenuation in gonadotropin β-subunit takes place. More importantly, change in gene expression was observed in adult hypothalamus, even although the animals were exposed to TCDD at fetal age (GD15). The genes reduced by TCDD include GnRH which plays a role in the expression of sexual behavior. This observation suggests that TCDD imprints the abnormality of gene